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published in

European Journal of Applied Physiology
2012

DOI (link to publisher)

[10.1007/s00421-012-2316-x](https://doi.org/10.1007/s00421-012-2316-x)

document version

Publisher's PDF, also known as Version of record

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citation for published version (APA)

Levels, K., de Koning, J. J., Foster Jr., C. C., & Daanen, H. A. M. (2012). The effect of skin temperature on performance during a 7.5-km cycling time trial. *European Journal of Applied Physiology*, 112(9), 3387-3395. <https://doi.org/10.1007/s00421-012-2316-x>

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The effect of skin temperature on performance during a 7.5-km cycling time trial

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Received: 7 September 2011 / Accepted: 6 January 2012 / Published online: 24 January 2012
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Abstract Aerobic exercise performance is seriously compromised in the heat. Possibly, a high skin temperature causes a rating of perceived exertion (RPE)-mediated decrease in exercise intensity. The purpose of this study was to determine the effect of skin temperature on power output during a 7.5-km cycling time trial. Thirteen well-trained male subjects performed a 7.5-km cycling time trial at 15°C and 50% relative humidity (CONTROL), with radiative heat stress during the time trial, and with (PRECOOL) or without (HEAT) precooling. Heat stress was applied by infrared heaters positioned in front of the cycle ergometer between 1.5 and 6.0 km. Skin, rectal, and pill temperature, power output, heart rate, and RPE were measured during the trial. Despite the lower mean skin temperature at the start of the time trial for PRECOOL compared to HEAT ($-2.1 \pm 0.7^{\circ}\text{C}$; $P < 0.01$) and CONTROL ($-1.8 \pm 0.6^{\circ}\text{C}$; $P < 0.05$), and a greater increase in mean skin temperature during the heat stress period for PRECOOL ($4.5 \pm 1.0^{\circ}\text{C}$) and HEAT ($3.9 \pm 0.8^{\circ}\text{C}$) than

for CONTROL ($-0.3 \pm 0.6^{\circ}\text{C}$; $P < 0.01$), no differences in power output were found between HEAT (273 ± 45 W) and CONTROL (284 ± 43 W; $P = 0.11$) and between HEAT and PRECOOL (266 ± 50 W; $P = 0.47$). Power output during the time trial was greater for CONTROL than for PRECOOL ($P < 0.05$). Additionally, no differences were observed in core temperature measures, HR, and RPE. Skin temperature does not affect the selection and modulation of exercise intensity in a 7.5-km cycling time trial.

Keywords Thermoregulation · Precooling · Heat stress · Exercise · Power output · Pacing strategy

Abbreviations

HR	Heart rate
PO	Power output
RPE	Rating of perceived exertion
RH	Relative humidity
T_{pill}	Pill temperature
T_{r}	Rectal temperature
T_{sk}	Mean skin temperature

Communicated by Narihiko Kondo.

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Introduction

The main goal of the human thermoregulatory system is to prevent unreasonable homeostatic disturbances by maintaining core body temperature within safe limits, both at rest and during exercise (Hardy 1961). During exercise, several heat dissipation mechanisms (e.g., radiation, convection, evaporation) act together to prevent an excessive rise in core temperature (Fortney and Vroman 1985). However, when metabolic heat production cannot be

compensated by heat dissipation (uncompensable heat stress), core body temperature may rise to critically high values. When an individual becomes hyperthermic, the activation of muscle motor units by the brain may be reduced, the sensation of fatigue occurs, and exercise performance can be seriously compromised (Gonzalez-Alonso et al. 1999; Nielsen et al. 1990; Nybo and Nielsen 2001).

Although a reduction in power output occurs with attainment of a high core temperature, studies on the effect of environmental heat stress on self-paced exercise performance have reported a reduction in power output, even when core temperature was not close to nominally critical values (Altareki et al. 2009; Ely et al. 2010; Kay et al. 2001; Marino et al. 2000, 2004; Tatterson et al. 2000; Tucker et al. 2004). In general, either early reductions in power output are observed or there is an inability to maintain a desired power output until the end of exercise. One possible explanation for the observed reductions in power output during self-paced exercise can be that exercise intensity is decreased to compensate for the environmental heat stress. A decrease in power output results in modulation of the rise in core temperature and thereby delays the attainment of a core temperature at which homeostasis could become compromised (Marino 2004; Tucker et al. 2006). This concept is one of the pillars of a recently developed model in which the regulation of exercise intensity during self-paced exercise is explained (Noakes et al. 2004, 2005; St Clair Gibson and Noakes 2004; Tucker 2009; Tucker and Noakes 2009). Within this model, it is proposed that the body functions as a complex system in which both anticipation and feedback are important in the regulation of exercise intensity during self-paced exercise. The anticipative component is reflected in the presence of a template of an acceptable rate of increase in the rating of perceived exertion (RPE) during exercise. This so-called RPE template is a theoretical construct based on expected exercise duration, previous experience, and physiological (e.g., skin temperature, core temperature) and psychological (e.g., motivation level) inputs before exercise (Tucker 2009). It describes an acceptable rate of increase in RPE during exercise that allows successful completion of the event while avoiding homeostatic disturbance. The feedback component includes integration of afferent information from different physiological systems (e.g., core and skin temperature, heart rate, and muscle glycogen content) during exercise. Information from these systems is used to constitute a conscious RPE that is matched with the RPE template. If the conscious RPE is different from the RPE template, the recruitment of skeletal muscle motor units, and thereby the power output, is adjusted (Tucker 2009).

Two factors that have been proposed as inputs for the anticipatory/feedback model of exercise regulation are skin temperature (Schlader et al. 2011b) and the rate of heat storage (Altareki et al. 2009; Marino 2004; Tucker et al. 2004, 2006). Cooling of the skin before the start of aerobic self-paced exercise has demonstrated beneficial effects on pacing pattern and performance (Kay et al. 1999; Ross et al. 2011). Schlader et al. (2011b) observed that skin temperature and thermal perceptions at the start of a 60-min cycling time trial were determinants of the selection of initial exercise intensity, with lower skin temperatures resulting in an increase in average power output. Although precooling seems to be beneficial for endurance exercise rather than for intermittent and short duration exercise (Marino 2002), lowering body temperature before exercise increases the ‘thermal reservoir’ (Nielsen 1994) and can possibly have positive effects on exercise performance. Tucker et al. (2006) instructed cyclists to cycle at a RPE value of 16 in cool (15°C), normal (25°C), and hot (35°C) conditions. They observed a greater rate of decline in power output in the hot condition than in cool and normal conditions. Additionally, they found a significantly greater rate of heat storage in the first 4 min of the trial in the hot condition. This greater rate of heat storage was a result of a higher skin temperature in the hot condition, as rectal temperature remained similar between conditions during this period. Since the calculation of rate of heat storage in this study has been invalidated (Jay and Kenny 2009), the influence of this parameter on the regulation of exercise intensity remains questionable. However, the initial difference in the skin temperature does appear to be an important factor for the regulation of exercise intensity. In summary, the skin temperature seems to be an important factor for both anticipation (selection of power output at the start of exercise) and feedback control (modulation of power output during exercise) of exercise intensity in self-paced exercise.

As mentioned earlier, changes in skin temperature mainly affect power output during mid-duration and long-duration exercise (Duffield et al. 2010; Ross et al. 2011; Schlader et al. 2011b; Tucker et al. 2006). If skin temperature is also an important input factor for the selection and modulation of power output during short-duration, self-paced exercise remains essentially uninvestigated. Therefore, the goal of this study was to examine the effects of a manipulation of skin temperature before and during exercise on performance and pacing pattern of a 7.5-km cycling time trial. We hypothesize that a lowering of skin temperature before the time trial increases initial power output, whereas an increase in skin temperature during the time trial decreases power output.

Methods

Subjects

Thirteen well-trained male cyclists participated in this study (Table 1). All participants gave written informed consent after receiving detailed information about the study and after being screened for known contraindications to exercise in the heat. The study was approved by the Research and Ethics Committee of TNO, The Netherlands.

Incremental exercise test and familiarization session

Subjects reported to the laboratory for preliminary testing consisting of an incremental exercise test and a 7.5-km familiarization time trial. The incremental exercise test was conducted on an electrically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands) in an environmental chamber (Weiss Enet, Tiel, The Netherlands) set at 15°C and 50% relative humidity (RH). After a 3-min warm-up period at a workload of 100 W, the test was started at 160 W and increased by 40 W every minute until volitional fatigue. During the test, heart rate was recorded at 5-s intervals (Polar s810i, Polar Electro Oy, Kempele, Finland). Respiratory gas exchange was measured breath by breath using a mask, covering both mouth and nose, connected to a gas analysis system (Jaeger Oxycon Pro, Viasys Healthcare, Hoechberg, Germany). $\dot{V}O_{2\text{Max}}$ was determined as the highest continuously recorded 30-s oxygen consumption. After the incremental test, subjects were given a 30-min recovery period before starting a 7.5-km practice cycling time trial to become familiar with the distance and procedure of the time trial. During the familiarization session no physiological parameters were measured. Apart from that, the trial was identical to the experimental trials.

Cycling time trials

Three experimental trials were performed in a balanced order and scheduled at least 2 days apart. Each trial consisted of a 30-min passive habituation period followed by a

3-min warming-up period and immediately after that a 7.5-km cycling time trial. The subjects were instructed to complete the time trial as fast as possible and were blind to performance measurements (power, cadence, and heart rate), but were given feedback about the covered distance. The three experimental conditions were: control (CONTROL), heat stress (HEAT), and precooling + heat stress (PRECOOL). In CONTROL, subjects remained seated in an environmental chamber at 20°C for 30 min. This period was followed by the cycling time trial at 15°C and 50% RH. HEAT was similar to CONTROL with the only difference being a period of extreme radiative heat stress that was imposed from 1.5 to 6.0 km during the time trial. The heat was generated by a panel consisting of 22 ceramic infrared heaters (FSR400, Elstein-Werk, Northeim, Germany), each generating a maximum of 400 W of heat. After the subject had completed 20% of the time trial, the heat panel was quickly positioned in front of the cycle ergometer, and after 80% of the distance was completed, the heat panel was quickly removed. This resulted in a distance of 4.5 km during which the subjects were exposed to the heat radiation. To maximize the effect of the sudden radiative heat exposure, subjects were wearing only cycling shorts and shoes and there was no airflow over the body. To quantify the heat radiation, a DRM Delta Radiometer (Helmut Hund GmbH, Wetzlar, Germany) was positioned at a distance from the heat panel that was equal to the distance from the subject's trunk to the heaters. PRECOOL started with a precooling period of 30 min. In this period, subject remained seated in an environmental chamber set at 10°C and were wearing a water-perfused suit (Med-Eng BCS4, Allen-Vanguard, Ottawa, Canada) connected to a thermal water bath (Tamson TLC-15, Tamson instruments, Bleiswijk, The Netherlands). The temperature of the water was maintained at 3°C. This suit covered the entire body, leaving only the area between mouth and eyes uncovered.

Measurements

Three hours before arrival at the lab, participants ingested a disposable core temperature capsule (Jonah, Hidalgo, Cambridge, UK) to measure intestinal temperature. After arrival at the lab, the subjects inserted a rectal thermometer (YSI401, Yellow Springs Instruments, Yellow Springs, OH, USA) 10 cm beyond the anal sphincter. Rectal temperature (T_r) was recorded every 10 min during the habituation period, and every 750 m during the time trial. To determine mean skin temperature (T_{sk}), four iButtons (DS1922L, Maxim Integrated Products Inc, Sunnyvale, CA, USA) were protected from direct radiation and taped to the skin (neck, right scapula, right shin, left hand). Mean skin temperature was calculated using Eq. 1 (ISO9886 2004).

Table 1 Descriptive statistics of the subjects ($n = 13$) including their responses at maximal exercise during the incremental exercise test (mean \pm SD)

Age (years)	24 \pm 4
Mass (kg)	76 \pm 7
Height (cm)	181 \pm 5
$\dot{V}O_{2\text{Max}}$ (ml kg ⁻¹ min ⁻¹)	59.3 \pm 3.6
HR _{Max} (beats min ⁻¹)	186 \pm 8
PO _{Max} (W)	388 \pm 30

$$T_{sk}(^{\circ}\text{C}) = 0.28 \times (T_{\text{neck}} + T_{\text{right scapula}} + T_{\text{right shin}}) + 0.16 \times (T_{\text{left hand}}) \quad (1)$$

Heart rate (HR) and pill temperature (T_{pill}) were recorded at 15-s intervals using the Hidalgo EquivitalTM Physiological Monitor system (Hidalgo, Cambridge, UK), and during the time trial, power output (PO) was measured per revolution. Data were averaged for every 750-m segment of the trial. The RPE was measured every 750 m on a 20-point scale (Borg 1982). Thermal perception and discomfort were measured on a 9-point and 5-point scale, respectively (Gagge et al. 1967), directly before the habituation period and directly before and after the time trial. The mass of the subjects was determined on a weighing scale (F300S, Sartorius, Göttingen, Germany) with a resolution of 1 g, directly before and after exercise.

Statistical analysis

Statistical analysis was performed in SPSS statistical software (SPSS 17.0, SPSS Inc., Chicago, IL, USA). Experimental condition (CONTROL, HEAT, PRECOOL) was the independent variable, whereas power output, pill temperature, rectal temperature, skin temperature, heart rate, and RPE were the dependent variables. The significance of effects of experimental condition on the dependent variables over time was determined using two-way ANOVA for repeated measurements, with two within-subject factors (experimental condition and distance completed). Post hoc analyses used Bonferroni correction to adjust for multiple comparisons. One-way ANOVAs were used to determine the significance of effects of experimental condition on final times, average power output, drift (slope) of heart rate, and thermal sensation and discomfort. The Pearson correlation coefficient was used to test for correlations between dependent measures. Statistical significance was set at the 5% level for each analysis. Values are reported as mean \pm SD.

Results

Precooling and heat stress intervention

In PRECOOL, the precooling intervention resulted in a pronounced decrease in T_{sk} (from 31.1 ± 0.6 to $29.9 \pm 0.6^{\circ}\text{C}$; $P < 0.05$), whereas no changes in T_r were observed (from 37.3 ± 0.4 to $37.2 \pm 0.4^{\circ}\text{C}$; $P > 0.05$). When the heat panel was placed in front of the subjects between 1.5 and 6.0 km (20–80% of time trial completion), the average radiation on the subjects' body during the time trial was $1,101 \pm 3 \text{ W m}^{-2}$ in HEAT and $1,102 \pm 3 \text{ W m}^{-2}$ in PRECOOL ($P > 0.05$).

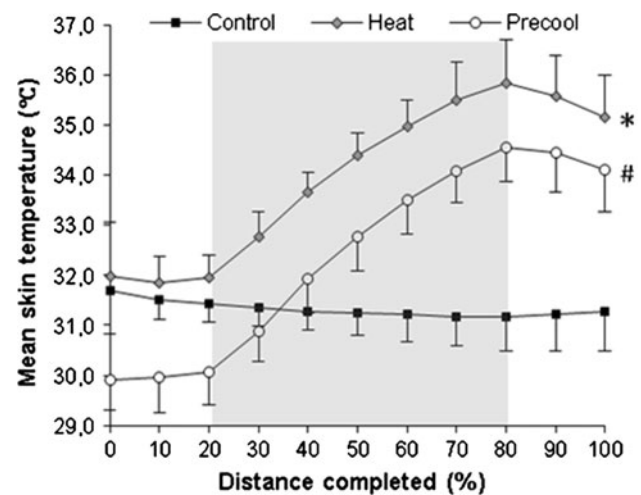


Fig. 1 Mean skin temperature during the cycling time trial for CONTROL, HEAT, and PRECOOL. The gray area represents the appliance of heat stress. Asterisk significant main effect with CONTROL and PRECOOL ($P < 0.05$); hash symbol significant main effect with CONTROL and HEAT ($P < 0.05$)

Mean skin temperature

At the start of the time trial, T_{sk} was $2.1 \pm 0.7^{\circ}\text{C}$ lower for PRECOOL than for HEAT ($P < 0.01$) and $1.8 \pm 0.6^{\circ}\text{C}$ lower than for CONTROL ($P < 0.05$). During the time trial, T_{sk} was $1.6 \pm 0.7^{\circ}\text{C}$ lower for PRECOOL than for HEAT ($P < 0.01$). There was a significant main effect present ($P < 0.01$) for T_{sk} , and from the start of the heat stress application, differences were observed among all the three conditions for each 10%-segment (except for the comparison between CONTROL and PRECOOL at 40% time trial completion; $P = 0.22$).

During the radiative heat stress period, the increase in skin temperature was significantly higher for HEAT ($3.9 \pm 0.8^{\circ}\text{C}$) and PRECOOL ($4.5 \pm 1.0^{\circ}\text{C}$) than for CONTROL ($-0.3 \pm 0.6^{\circ}\text{C}$) ($P < 0.01$ for both differences). At the end of the heat stress period, T_{sk} was $35.8 \pm 1.0^{\circ}\text{C}$ for HEAT, $34.6 \pm 0.8^{\circ}\text{C}$ for PRECOOL, and $31.2 \pm 1.0^{\circ}\text{C}$ for CONTROL ($P < 0.01$). From the start until the end of the radiative heat stress (20–80% of distance completed), skin temperature was higher during every 10%-segment for HEAT than for CONTROL. T_{sk} during the cycling time trial is shown in Fig. 1.

Core temperature

Both T_r and T_{pill} increased gradually during the cycling time trial (0.04 ± 0.02 and $0.05 \pm 0.02^{\circ}\text{C min}^{-1}$, respectively; Fig. 2), but no differences were observed in both T_r and T_{pill} between the conditions ($P = 0.81$ and $P = 0.86$ for T_r and T_{pill} , respectively). The highest observed T_r was $37.6 \pm 0.3^{\circ}\text{C}$, whereas the highest T_{pill} was $37.8 \pm 0.2^{\circ}\text{C}$, both at completion of the time trial in HEAT.

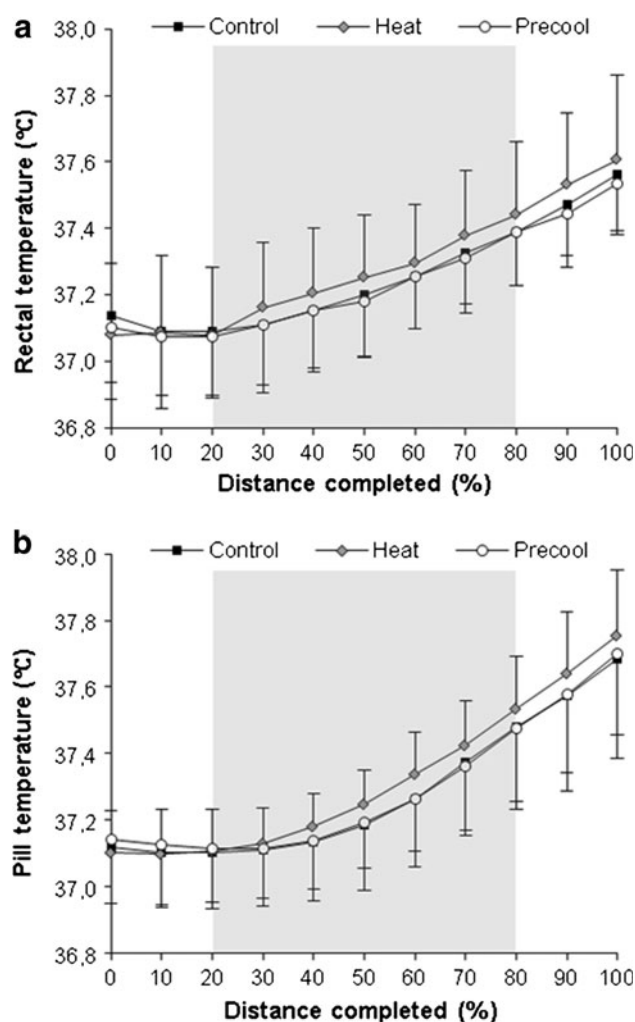


Fig. 2 Rectal (a) and pill (b) temperature during the cycling time trial for CONTROL, HEAT, and PRECOOL. The gray area represents the appliance of heat stress

Time trial performance and power output

Time to completion of the 7.5-km time trial was not different for CONTROL (684 ± 95 s), HEAT (725 ± 111 s), and PRECOOL (739 ± 130 s; $P = 0.47$). Mean power output over the time trial was significantly greater in CONTROL than in PRECOOL (284 ± 43 and 266 ± 50 W, respectively; $P < 0.05$), but no significant differences were observed between HEAT (273 ± 45 W) and CONTROL ($P = 0.11$) and between HEAT and PRECOOL ($P = 0.47$).

For the three conditions, average PO for every 10% segment of the distance completed in the time trial is shown in Fig. 3. Although mean PO of the entire time trial was greater in CONTROL than in PRECOOL, no significant differences were observed between the conditions in separate 10% segments. For each condition, the pacing profile was slightly negative with a marked increase in the final 10% of the time trial in which the average PO was

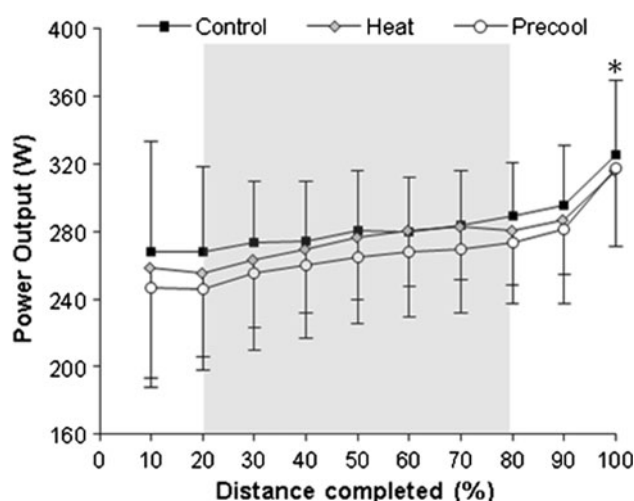


Fig. 3 Mean power output during each 10% of completed distance of the time trial for CONTROL, HEAT, and PRECOOL. The gray area represents the appliance of heat stress. The first 300-m of the time trial were not included in the calculation of mean power output during the first 750-m. Asterisk significantly higher PO than other 10% segments for all the conditions ($P < 0.05$)

significantly higher than in the previous intervals for all the conditions ($P < 0.05$). No correlation was observed for PO and T_{sk} ($P = 0.11$), whereas a weak inverse correlation was found for change in T_{sk} during the heat stress period (20–80% of time trial completion) and the average PO during this period ($r = -0.37$; $P = 0.02$).

Heart rate and RPE

No significant main effect was found for HR ($P = 0.07$), but at the end of the heat stress period (80% of distance completed), HR was significantly lower for CONTROL (164 ± 8 bpm) than for HEAT (172 ± 6 bpm; $P = 0.02$) and for PRECOOL (171 ± 5 bpm; $P = 0.02$) (Fig. 4). Also, the drift in HR during the heat stress period was higher for HEAT and PRECOOL than for CONTROL (3.9 ± 0.7 and 3.8 ± 0.8 vs. 3.2 ± 1.1 bpm min^{-1}). The maximum heart rate was achieved at the end of the time trial and was similar for CONTROL, PRECOOL, and HEAT (175 ± 9 , 178 ± 5 , and 180 ± 7 bpm, respectively; $P = 0.34$).

RPE values increased similarly for the three conditions during the time trial ($P = 0.937$), and the average value at the end of the time trial was 18.7 ± 0.9 for CONTROL, 18.5 ± 1.1 for HEAT, and 18.7 ± 0.5 for PRECOOL (Fig. 5).

Thermal sensation and comfort

Thermal sensation was significantly lower before start of the time trial in PRECOOL (-1.9 ± 0.5) than in HEAT (-0.3 ± 1.3 ; $P = 0.01$), and similar to CONTROL (-0.9 ± 1.2 ; $P = 0.24$). These results were accompanied

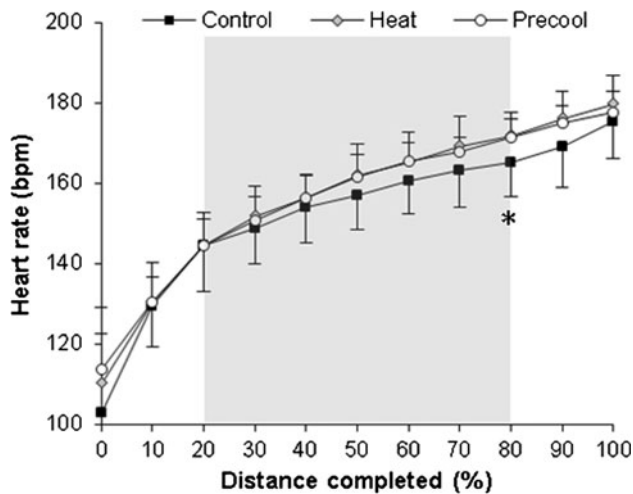


Fig. 4 Heart rate during the cycling time trial for CONTROL, HEAT, and PRECOOL. The gray area represents the appliance of heat stress. Asterisk significantly lower heart rate for CONTROL than for HEAT and PRECOOL

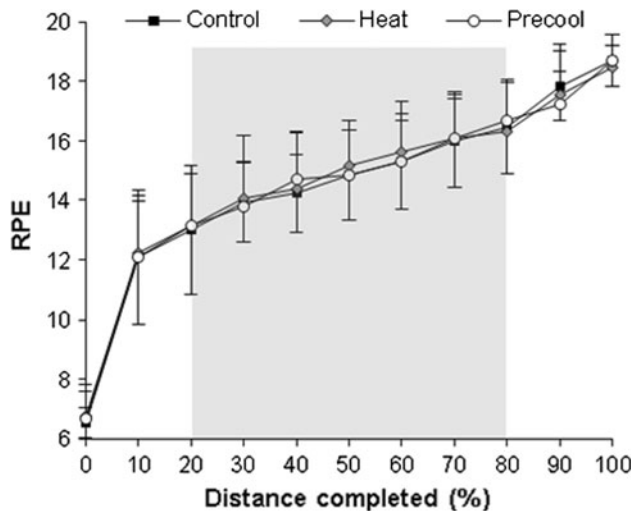


Fig. 5 Rating of perceived exertion during the cycling time trial for CONTROL, HEAT, and PRECOOL. The gray area represents the appliance of heat stress

by a higher value for thermal discomfort in PRECOOL (2.2 ± 0.7) than in HEAT (1.4 ± 0.5 ; $P = 0.02$), and similar values for CONTROL (1.6 ± 0.7 ; $P = 0.08$ for difference with PRECOOL). At the completion of the time trial, no differences were found between the conditions in either thermal sensation or thermal discomfort ($P = 0.86$ for thermal sensation and $P = 0.95$ for thermal discomfort).

Discussion

The purpose of this study was to evaluate the effect of a manipulation of skin temperature before and during a

7.5-km cycling time trial on pacing pattern and performance. The main outcome was that skin temperature does not affect the selection and modulation of exercise intensity in this type of short-duration exercise. Therefore, we have to reject our hypothesis that a lowering of skin temperature before the time trial increases initial power output and that an increase in skin temperature during the time trial decreases power output.

The experimental set-up used in this experiment was similar to the set-up used by Johnson et al. (2009), who investigated the effects of the sudden application of hypoxia on power output during a 5-km cycling time trial. The protocol allowed us to investigate the importance of skin temperature as anticipatory signal at the start of exercise, and as afferent (thermophysiological) feedback during exercise. Because of the abrupt change in heat stress, also the temporal aspect of the response in power output could be investigated.

In this study, the average skin temperature at the start of the cycling time trial was $2.1 \pm 0.7^\circ\text{C}$ lower for PRECOOL than for HEAT. Therefore, the precooling intervention was successful. No differences were found in rectal and pill temperature, heart rate, RPE, and respiratory measures. Interestingly, initial exercise intensity was similar for these conditions (247 ± 79 W for PRECOOL and 258 ± 71 W for HEAT), despite the difference in skin temperature. These observations imply that for this type of exercise (short-duration, self-paced), skin temperature at the beginning of exercise is not an important factor for the selection of initial exercise intensity. In a recent study, Schlader et al. (2011b) examined the effect of skin temperature at the start of a 60-min cycling time trial on power output. They observed that the average power output was higher when subjects started with a low skin temperature and the skin was warmed during the time trial, than when they started with a high skin temperature and were cooled during the time trial. The higher average power output was mainly a result of the higher initial power output in the cold-to-hot trials. They concluded that, in contrast with our study, skin temperature (and accompanying thermal perception) at the beginning of the exercise was an important input signal for the selection of initial exercise intensity. This suggests that the signal of skin temperature is interpreted differently based on the anticipated duration of the event.

Another interesting observation in this study was the lower average power output during the cycling time trial for PRECOOL than for CONTROL. Our original hypothesis that precooling could increase the ‘thermal reservoir’ and thereby improve performance (Marino 2002; Nielsen 1994), therefore, needs to be rejected for performances shorter than 12 min. Although skin temperature at the beginning of the exercise was markedly reduced in

PRECOOL (Fig. 1), changes in power output (Fig. 3) were not observed. Since the applied method of precooling was whole-body cooling, also the legs were precooled. Therefore, a possible explanation for the reduction in average PO in PRECOOL could be a lower muscle temperature, leading to a greater contribution of anaerobic glycolysis at the beginning of exercise (Beelen and Sargeant 1991) and possibly a less effective muscle performance (Bennett 1984).

In this experiment, we showed that the application of radiative heat stress during exercise in PRECOOL and HEAT resulted in an increase in mean skin temperature during the time trial, whereas mean skin temperature slightly decreased over time in CONTROL (Fig. 1). Since both RPE and power output during the time trial were similar for CONTROL and HEAT, it can be concluded that in short-duration, self-paced exercise, skin temperature during exercise does not seem to be an important feedback signal for the RPE-based regulation of exercise intensity. This finding is supported by the weak inverse correlation between skin temperature and power output during the heat stress period and appears to be in disagreement with previous studies (Altareki et al. 2009; Tucker et al. 2006). Tucker et al. (2006), for example, observed a significantly higher rate of heat storage in the first 4 min of a RPE-clamped cycling trial in the heat (35°C) compared to cool (15°C) and normal (25°C) temperatures. Because rectal temperature was similar for the three conditions, the higher rate of heat storage was a result of a higher skin temperature. Since the rate of decline in exercise intensity was significantly higher in the heat, it can be deduced that afferent feedback regarding skin temperature affected the selection and modulation of exercise intensity. However, one important difference between the study of Tucker and our study is the duration of the time trial. In Tucker's study, the trial duration varied between 34.0 and 50.2 min depending on experimental condition, whereas in our study the distance of the time trial was (only) 7.5 km and the trial duration was ~12 min, which is at least three times shorter than in Tucker's study. The short duration of the time trial and the associated limited rise in core temperature in our study may explain the discrepancy in results between our study and previous studies. Traditionally, core temperatures of 39.0°C or higher are associated with fatigue during exercise in the heat (Cheung and Sleivert 2004; Nybo 2007). In short-duration exercise, however, the rise in core temperature is limited and probably exercise-limiting hyperthermia does not occur. However, recent studies show that aerobic performance in the heat can be reduced without marked hyperthermia (Altareki et al. 2009; Ely et al. 2010). In these studies, core temperature values were ~0.5°C higher than in our study, but still well below the values that are generally associated with fatigue.

Although the short duration of the time trial (and the associated limited rise in core temperature) could explain the absence of visible reductions in exercise intensity during the time trial, we suggest that during this type of short-duration, self-paced exercise, thermal information is relatively unimportant for the selection and adaptation of exercise intensity. We concur that the importance of signals in the regulation of pacing strategy differs with the distance/duration of the exercise bout (de Koning et al. 2011). In De Koning's study, the relative importance of physiological feedback signals in the regulation of pacing strategy was compared with the monitoring of different gauges by the driver of a race car during a race. One particular gauge is more important during short races, whereas another gauge is more important during long races. It appears that (the increase in) skin temperature is not an important physiological signal during a short-duration exercise bout. Since the radiative heat stress applied in this study elicited relatively large changes in skin temperature, we do not expect that an even greater increase in skin temperature would elicit RPE-mediated changes in power output. For future studies, it would be of interest to investigate from which distance/duration, a change in skin temperature does become an important feedforward and feedback signal. This signal seems to play a minor role in a cycling time trial of 7.5 km and duration of ~12 min, whereas it is important in cycling time trials of ~35–50 (Tucker et al. 2006) and 60 min (Schlader et al. 2011b).

Not only skin temperature, but also thermal perceptions have been suggested as relevant signals for the selection and modulation of exercise intensity (Schlader et al. 2011a), possibly by affecting the motivation to continue exercise in the heat (Cotter et al. 2001). In our study, thermal sensation was lower and thermal discomfort was higher before the start of the time trial in PRECOOL compared to HEAT. At the completion of the time trial, no differences were found between the conditions in either thermal sensation or thermal discomfort. The lower thermal sensation and the higher thermal discomfort before the start of the time trial could have resulted in a downwards shift of power output during exercise. Unfortunately, we did not record ratings of thermal sensation and discomfort during exercise, so the relationship between changes in thermal perception during exercise and power output remains unclear. For future studies, we would recommend recording thermal perceptions during exercise as these signals appear to be especially relevant for the selection and modulation of exercise intensity (Schlader et al. 2011a).

In this study, mean skin temperature was determined using four iButtons. Using more locations to measure skin temperature would have increased the measurement accuracy (Mitchell and Wyndham 1969), but relatively more iButtons would then have been in close contact with the

radiation of the heat panel. Although the iButtons were adequately shielded from the radiation, this might possibly have caused an overestimation of mean skin temperature, which we wanted to prevent. Moreover, since the subjects did not perceive their skin to be cold (Livingstone et al. 1987), we believe the measured change in skin temperature reflects the actual change in skin temperature caused by the interventions, and that using only four measurement locations did not limit our findings. To determine core body temperature, both a rectal thermistor and a disposable core temperature capsule were used. Since both rectal and intestinal temperature react slower to a changing body temperature than esophageal temperature (Byrne and Lim 2007), using these methods may limit our findings. However, using esophageal temperature as an index of core body temperature was not an option due to health risks associated with the esophageal thermistor and the extreme radiative heat.

Conclusion

Changes in starting skin temperature and skin temperature during a 7.5-km cycling time trial do not affect performance and pacing pattern. Therefore, we suggest that skin temperature is relatively unimportant for the anticipatory selection of initial exercise intensity and RPE-mediated modulation of power output during short-duration, self-paced exercise.

Acknowledgments The authors express their thanks to the participants for their time and effort.

Conflict of interest The authors declare that they have no conflict of interest.

Ethical standards The experiments conducted in this study comply with the current laws of the Netherlands.

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